Limits: **Publication Date to 2001**

1: Acta Neurol Scand. 1976 Jan;53(1):39-50.

Circulating IgE, allergy and multiple sclerosis. Serum levels of IgE, other immunoglobulins and complement (C's) in patients with multiple sclerosis in exacerbation and other neurologic diseases.

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Serum levels of IgE, other immunoglobulins and C'3 were measured in 36 MS patients, and the results compared with those of 40 age- and sex-matched patients hospitalized on Neurology Service. Diagnoses among controls included cerebral infarction, cervical spondylosis, nonmigrainous headache, seizure disorders and peripheral neuropathy. Six patients in the MS group and seven in the non-MS group had a past history of allergy to food, drugs, dust or other substances. IgE levels were measured by double-antibody radioimmunoassay. Other immunoglobulins (G, M, D and A) and C'3 were quantitated by Mancini's method. Results indicate that the median IgE and C'3 levels of MS patients were slightly lower than those for non-MS subjects. Concentrations of other immunoglobulins were similar for the two groups. CSF samples from 12 MS and five non-MS patients were studied and none of these contained measureable (greater than 7.5 U/ml) IgE. In view of: 1) recent reports describing mast cells in MS plaques, 2) the suggested role of biogenic amines in the pathogenesis of demyelinating diseases, and 3) because IgE exists in free and cell bound state, it is suggested that tissue surveys of MS plaques for IgE deposits similar to those seen in glomeruli in nephrotic syndromes may provide a clue to the pathogenesis of MS.

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1: J Invest Dermatol. 1990 Jun;94(6 Suppl):49S-52S. Related Articles, Links

Fc receptors for IgE and interleukin-4 induced IgE and IgG4 secretion.

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IgE binds to two types of Fc receptors, called Fc epsilon R1 (or high-affinity Fc epsilon R) and Fc epsilon R2 (or low-affinity Fc epsilon R). The Fc epsilon R1 is composed of four polypeptide chains, one alpha, one beta, and two gamma chains. The alpha chain contains the IgE binding site and is a member of the immunoglobulin supergene family. The Fc epsilon R2, also called CD23, consists of one polypeptide chain which shows homology to animal lectin receptors. Fc epsilon R1 are expressed on mast cells and basophils. Crosslinking of the Fc epsilon R1 induces immediate release of mediators of inflammation such as histamine and leukotrienes and delayed secretion of interleukins 4, 5, and 6. Fc epsilon R2 are expressed on resting mu delta + B cells, monocytes/macrophages (M phi), eosinophils, and platelets but rarely on T cells. Interleukin-4 upregulates Fc epsilon R2 expression on B cells and M phi. The functions of Fc epsilon R2 on the different cell types are not fully established and are controversial. Fc epsilon R2 on M phi, eosinophils, and platelets mediate cytotoxicity to schistosomules, enhance phagocytosis, and induce the release of granule enzymes. However, M phi from patients with atopic dermatitis expressing significantly more Fc epsilon R2 than M phi from normals do not release more leukotriene C4, prostaglandin E2, or beta-glucuronidase after incubation with aggregated IgE than normal monocytes. Furthermore, aggregated IgG1 is much more efficient than IgE in inducing mediator release from M phi and IgG1 antibodies are not known to induce immediate-type hypersensitivity reactions. Therefore, definitive proof that Fc epsilon R2 are involved in the pathogenesis of allergic disorders is still lacking. IL-4 appears to play a central role in immediate-type hypersensitivity. It induces human B cells to secrete IgE and IgG4, Ig isotypes typical for antibodies to helminthic parasites and allergens. IL-4 stimulates mast cell growth and

upregulates Fc epsilon R2 expression. Interferon-gamma and IL-2 inhibit the IL-4-induced IgG4 and IgE secretion. Whether the abnormally high IgE antibody production in atopic patients is the result of overproduction of IL-4 or deficient IFN-gamma/IL-2 production is presently unknown.

Publication Types:

- Review
- Review, Tutorial

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1: Cell Immunol. 1996 Jul 10;171(1):111-9.

ELSEVIER FULL-TEXT ARTICLE

Antigen-driven peripheral immune tolerance: suppression of experimental autoimmmune encephalomyelitis and collagen-induced arthritis by aerosol administration of myelin basic protein or type II collagen.

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Antigen-driven tolerance is an effective method of suppressing cell-mediated immune responses. We have previously demonstrated that exposure of gut-associated lymphoid tissue to myelin basic protein (MBP) via oral administration suppresses experimental autoimmune encephalomyelitis (EAE). To further study presentation of antigen to the immune system by mucosal surfaces as a method of antigen-driven tolerance, the effect of inhalation of MBP was investigated. MBP was given as an aerosol to Lewis rats on Days -10, -7, -5, and -3 prior to immunization with MBP in Freund's adjuvant and on Days 0, 2, and 4 following immunization. Aerosolization of MBP completely abrogated clinical EAE in 100% of treated rats. Central nervous system inflammation and delayed-type hypersensitivity and antibody responses to MBP were also significantly reduced in aerosol-treated animals. Aerosolization of histone, a basic protein of similar weight and charge as MBP, had no effect. Disease was also suppressed with one aerosol treatment on Day -3 or by administering MBP nasally. Aerosolization was more effective than oral administration of MBP over a wide dose range (0.005-5 mg). Splenic T cells isolated from animals postaerosolization adoptively transferred protection to naive animals immunized with MBP. Aerosolization of MBP to animals with relapsing EAE after recovery from the first attack decreased the severity of a subsequent attack. Aerosol and oral MBP were equally effective at suppressing the in vitro immune response as measured by proliferation and interferon-gamma production. We then tested aerosolization of a different autoantigen in a different disease model and found that aerosolization of type II collagen was effective in suppressing collagen-induced arthritis. Thus, aerosolization of an autoantigen is a potent method to downregulate

an experimental T cell-mediated autoimmune disease and suggests that exposure of antigen to lung mucosal surfaces preferentially generates immunologic tolerance.

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